

ment and no other respiratory organisms were grown we attempted to identify the organism and carried out sensitivity tests. The organism was resistant to penicillin, ampicillin, cephalixin, fusidic acid, and co-trimoxazole, and was sensitive to tetracycline, lincomycin, and gentamicin. Treatment was changed to lincomycin 500 mg by mouth thrice daily. Before this treatment was started a third specimen of sputum and second specimen of blood were obtained for culture. The sputum specimen again grew the same organism, but blood cultures showed no growth after two weeks' incubation. She became clinically well within five days after the changed treatment. The antibiotic was continued for a total of seven days. Two days later a further specimen of sputum was examined. This was not purulent and did not grow the organism or any known respiratory pathogen. She was discharged and was well at follow-up two weeks later.

Bacteriology—Overnight incubation at 37°C produced whitish raised colonies of about 0.5 mm in diameter. Gram-staining showed large non-sporing Gram-positive rods. The organism grew aerobically as well as anaerobically, but slightly better in the presence of carbon dioxide. It was catalase and oxidase negative and non-motile. Similar characteristics were observed in all three cultures. The organism was provisionally named as a *Lactobacillus* spp and later fully identified by the Central Public Health Laboratory, London, as *Lactobacillus casei* ss *rhamnosus*.

Comment

That this patient's chest infection was caused by a lactobacillus was proved by the following: presence of symptoms and repeated isolation of the same organism from sputum in pure growths; absence of clinical response to cephalixin, to which the organism was insensitive, and clinical and bacteriological cure after treatment with lincomycin, to which the organism was sensitive.

To my knowledge this is the first reported case of a lactobacillus producing chest infection in the absence of septicaemia. Because of their colonial and Gram-staining characteristics lactobacilli are easily mistaken for diphtheroids. Like these, lactobacilli, including the species isolated in this case, may also occur as normal flora in the mouth and gastrointestinal and female genital tracts. The other natural habitats of lactobacilli are milk and milk products.⁵ The clinical importance of isolation of such organisms from non-sterile specimens—for example, sputum—could be established only by repeated isolation in pure growths and by matching clinical cure with negative bacteriology.

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Faecal peritonitis after laxative preparation for barium enema

The use of purgatives in the routine preparation of the colon is widespread in radiological practice in Britain.¹ Evaluation of a double-contrast enema in particular depends on meticulous cleansing of the bowel in order to distinguish small lesions.²

We report two cases in which administration of X-Prep (Napp Laboratories Ltd), a concentrated sennoside, was followed by fatal colonic perforation.

Case reports

CASE 1

A 71-year-old man had a two-month history of intermittent diarrhoea and rectal bleeding. He was anorexic and had lost 2 kg in weight over the preced-

ing two months but had not complained of abdominal pain. He was referred for outpatient barium enema and was prescribed X-Prep. He took 71 ml X-Prep at 5 pm on the evening before the planned investigation. An initial small bowel movement was followed four hours later by colicky lower abdominal pain, which gradually increased in severity.

He was admitted to hospital the next morning with generalised abdominal tenderness and guarding. At 6 am he developed tachycardia of 120 beats/min, blood pressure fell to 70/50 mm Hg, and he became increasingly dyspnoeic. An erect abdominal radiograph showed free gas under the left diaphragm. He was resuscitated with intravenous fluids and antibiotics. Laparotomy disclosed faecal peritonitis. Diverticular disease of the descending colon was present but no intraluminal narrowing or identifiable perforation. The abdomen was drained and he was admitted to the intensive therapy unit for intermittent positive-pressure ventilation. He developed acute renal failure and irreversible peripheral ischaemia of the leg and died two days later. Permission for necropsy was refused.

CASE 2

A 65-year-old man had a two-month history of diarrhoea and rectal bleeding. On rectal examination a large tumour was palpable, and he was admitted for urgent investigation and treatment. He received a standard 71 ml dose of X-Prep on the day before a scheduled barium enema. Later that evening he experienced colicky abdominal pain which initially settled, but at 5.45 am the next morning he developed generalised abdominal pain with rigidity and rebound tenderness.

At laparotomy he had faecal peritonitis secondary to a 10 cm tear at the rectosigmoid junction. There was gross intraperitoneal and retroperitoneal soiling. The torn area of colon was resected and the upper portion brought out as an end colostomy. The peritoneal cavity was drained with several polyethylene tube drains. He was admitted to the intensive therapy unit for intermittent positive-pressure ventilation. He required dopamine and later isoprenaline to maintain his blood pressure. Despite initial improvement he died 14 days later of *Clostridium welchii* septicaemia. At necropsy there was no evidence of spread of the rectal tumour, which did not occlude the rectal lumen.

Comment

Senna is obtained from an Arabian shrub, and modern preparations can be standardised. X-Prep contains 142 mg sennosides A and B in each 71 ml dose. This is almost six times the maximum recommended daily dose of 24 mg.³

Most radiology departments consider X-Prep to be contraindicated in ulcerative colitis, Crohn's disease, and acute diverticulitis because of the risk in subjecting a friable colon to a period of intensive peristalsis. In neither of these cases was there clinical evidence of acute inflammation or obstruction of the colon. When X-Prep is used the development of abdominal pain should raise suspicion of colonic perforation.

No detailed information is available on how many patients in Britain receive X-Prep before a barium enema. In the area of the Greater Glasgow Health Board, which has a population of 990 000, about 12 400 barium enemas are performed each year. A total of 10 000 patients are given X-Prep before the procedure and are consequently at risk of developing this complication. In addition, several hospitals in the region use the drug before intravenous pyelography.

Perforation of the colon is a recognised complication of barium enema itself⁴ but has only once been reported after the use of X-Prep.⁵ The Committee on Safety of Medicines has had no previous notification of this complication in Britain.

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